REVIEW

Acute aortic syndrome: pathology and therapeutic strategies

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Acute aortic syndrome (AAS) describes the acute presentation of patients with characteristic ''aortic pain'' caused by one of several life threatening thoracic aortic pathologies. These include aortic dissection, intramural haematoma, penetrating atherosclerotic ulcer, aneurysmal leak, and traumatic transection. AAS heralds imminent aortic rupture. Highlighting acute aortic pathology as an AAS is therefore important to encourage prompt recognition of this condition and avoid diagnostic delays. The management of AAS remains a therapeutic challenge. The traditional surgical approach to acute ''type B'' (descending thoracic) aortic pathology is unsatisfactory with high morbidity and mortality. Endovascular aortic stent grafts now represent an alternative minimally invasive approach in these patients who are often poor surgical candidates. Studies show endovascular repair to be technically feasible with fewer complications. This review discusses AAS pathology and in particular assesses the current role for endovascular aortic repair in its treatment.

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Acute aortic syndrome (AAS) describes the acute presentation of patients with one of several life threatening thoracic aortic pathologies. These include aortic dissection (AD), intramural haematoma (IMH), penetrating atherosclerotic ulcer (PAU), aneurysmal leak, and traumatic transection (box 1).[1–3]

AAS is characterised by typical presenting features (box 2) and a predictable patient profile (table 1). Classically (in non-traumatic cases) elderly patients present with characteristic ''aortic pain'' on a background of severe hypertension and comorbidity including coronary artery disease, diabetes, and renal insufficiency.[2 3] Aortic pain has been variously described as severe, ripping, and migratory chest pain that may radiate anteriorly to the neck or posteriorly between the scapula when it affects the ascending or descending aorta respectively.[1 3 4] It has been suggested that in distinction to the gradual increasing intensity of dull cardiac pain, AAS has an abrupt onset with maximal intensity often at the time of onset.[5]

The need to consider and highlight acute thoracic pathology as an AAS is clear. In an ageing morbid population and with modern imaging techniques it is becoming a more commonly encountered clinical phenomenon. Furthermore, thoracic aortic pathology can be a difficult diagnosis to make. Clinical findings are often absent,[4] the chest radiograph may be normal, and symptoms may be confused with acute myocardial infarction, which should be excluded. An AAS therefore encourages prompt recognition of symptoms heralding an unstable phase in these disease processes indicating imminent aortic rupture. This will hopefully expedite recognition and avoid diagnostic delays.

The comparatively new concept of an acute aortic syndrome also reflects recent advances in the management of thoracic aortic pathology. Aortic pathology is often unsuitable for conservative medical treatment and many patients are also poor surgical candidates. Over the past decade dramatic improvements in aortic imaging has led to a better understanding of thoracic aortic pathology. As a result minimally invasive endovascular aortic repair is now increasingly being undertaken.

The aim of this review is therefore to provide an overview of AAS pathology and in particular to discuss and assess the current role for endovascular aortic repair in its treatment.

PATHOLOGY CAUSING ACUTE AORTIC SYNDROME

All disorders giving rise to AAS can be distinguished in terms of their aetiology and radiological appearance. There is however considerable overlap with the possibility of progression from one pathological process to another. They also share a common classification system. Figure 1 shows the Stanford classification that was originally applied to aortic dissection. This defines aortic disease according to site that broadly correlates with management. Type A disease by definition affects the ascending aorta (and aortic arch) and type B, the descending aorta (distal to the origin of the left subclavian artery).

Aortic dissection

In aortic dissection the primary pathology is an intimal tear that penetrates the aortic media. This entry tear occurs at sites of greatest wall tension. Usually within a few centimetres of the aortic valve on the right lateral wall of the ascending aorta (type A dissection) or close to the site of the ligamentum arteriosum in the descending aorta where the aorta is fixed.[2 3 7] Blood at high pressure then splits or dissects the

Abbreviations: AAS, acute aortic syndrome; AD, aortic dissection; IMH, intramural haematoma; PAU, penetrating atherosclerotic ulcer; CT, computed tomography; MRI, magnetic resonance imaging

Box 1 Thoracic pathology included in acute aortic syndrome

- Dissection
- Intramural haematoma
- Penetrating atherosclerotic ulcer
- Aneurysm leak
- Trauma transection

Box 2 Clinical presentation of acute aortic syndrome[1 3 4]

- Chest pain—severe, acute, ripping, migratory
- Radiation of pain to anterior chest or neck—when pathology in ascending aorta
- Radiation of pain to back—when pathology in descending aorta
- Difference in upper extremity blood pressure
- Pulse deficits
- End organ ischaemia

Note: Physical findings are often absent

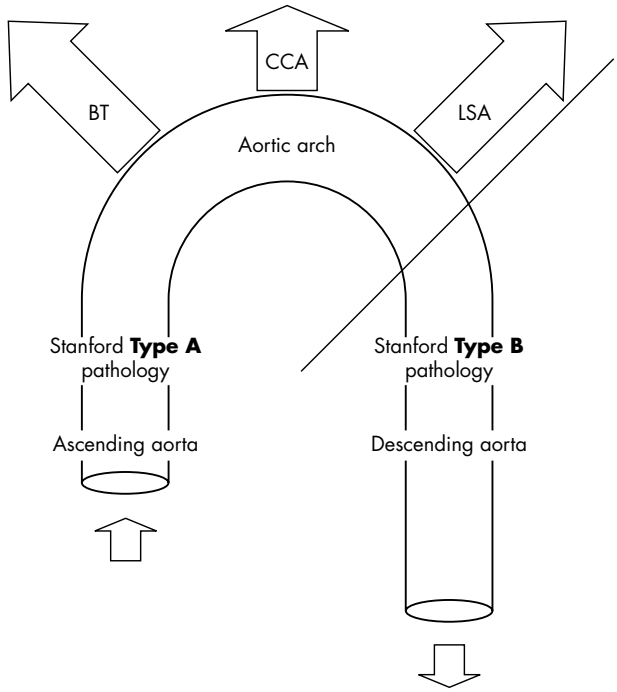

Figure 1 Schematic diagram of the Stanford classification of thoracic aortic pathology. See text for explanation. BT, brachiocephalic trunk; CCA, common carotid artery; LSA, left subclavian artery.

media to form a false channel or lumen that runs alongside the true lumen. A further re-entrance tear allows blood to circulate through the false lumen (see fig 2A and B).[1] Ischaemic symptoms occur when the dissection flap occludes an aortic branch. Interestingly most dissections are not associated with atherosclerosis. In fact fibrosis and calcification may limit the progression of dissection.[7 8]

PAU

PAU is a focal defect or lesion occurring at the site of intimal atherosclerotic plaques (see fig 2C). Patients therefore tend to be older with greater cardiovascular comorbidity. It occurs most commonly in the descending aorta, which may reflect more atheromatous disease here.[11] Progressive intimal erosion can lead to pulsatile blood entering the media resulting in haemorrhage. Formation of IMH (because of erosion of aortic vasa vasorum by the ulcer) or dissection can both occur. Further penetration to adventitia can cause pseudoaneurysm, aneurysm formation or even rupture.[2 4 6 7 9] PAU also has the highest rate of aortic rupture (up to 42%),[8] when compared with IMH or dissection.

IMH

IMH is thought to account for 10% to 30% of all cases of acute aortic syndrome.[5 10] It is a variation of dissection where blood collects within the aortic media without the presence of an intimal flap.[1 4] This occurs because of rupture of the nutrient vasa vasorum that lies in the media or from haemorrhage within an atherosclerotic plaque.[11] It may therefore be related to hypertension or blunt trauma or may arise from a PAU.[2] IMH may resolve spontaneously or progress and increase in size. Unlike dissection, IMH occurs much closer to the adventitia (outer layer or the arterial wall), which explains its higher rate of rupture (35%)[8 12] compared with dissection. It may also extend towards the aortic lumen and progress to dissection.[1 6] Pleural, pericardial, and mediastinal haemorrhage have also been described and are related to increased aortic wall permeability.[3] Progress may be sudden, unpredictable, and will present as an AAS.

Table 1 Risk factors and comorbidity related to acute aortic syndrome[2–4 6]

Risk factors	Comorbidity
Increasing age	Coronary artery disease
Chronic hypertension	Cerebrovascular disease
Atherosclerosis	Peripheral artery disease
Collagen vascular disorders (for example, Marfans)	Renal disease
Blunt thoracic trauma	Diabetes
Previous aortic valve surgery	Polytrauma

Aortic aneurysm leak

AAS may be related to the acute expansion of an aneurysm. According to LaPlaces Law progressive vessel dilatation results in increasing vessel wall tension and therefore further more rapid dilatation and risk of rupture.[13] Aneurysms may be associated with the progression of dissection, IMH, or PAU because of both weakening of the aortic wall and persistent hypertension[2] (see fig 2D).

Traumatic transection

Traumatic transection occurs as a result of rapid deceleration forces or direct trauma with most resulting in immediate death. The most commonly injured site is just distal to the left subclavian artery at the aortic isthmus where the aorta is fixed by the ligamentum arteriosum.[7] A minority may present with an incomplete rupture resulting in dissection, IMH, or pseudo-aneurysm formation.[2]

IMAGING IN ACUTE AORTIC SYNDROME

Imaging plays a vital part in diagnosing AAS. Although a common screening tool in the emergency department, the chest radiograph has a limited role. It has poor sensitivity for

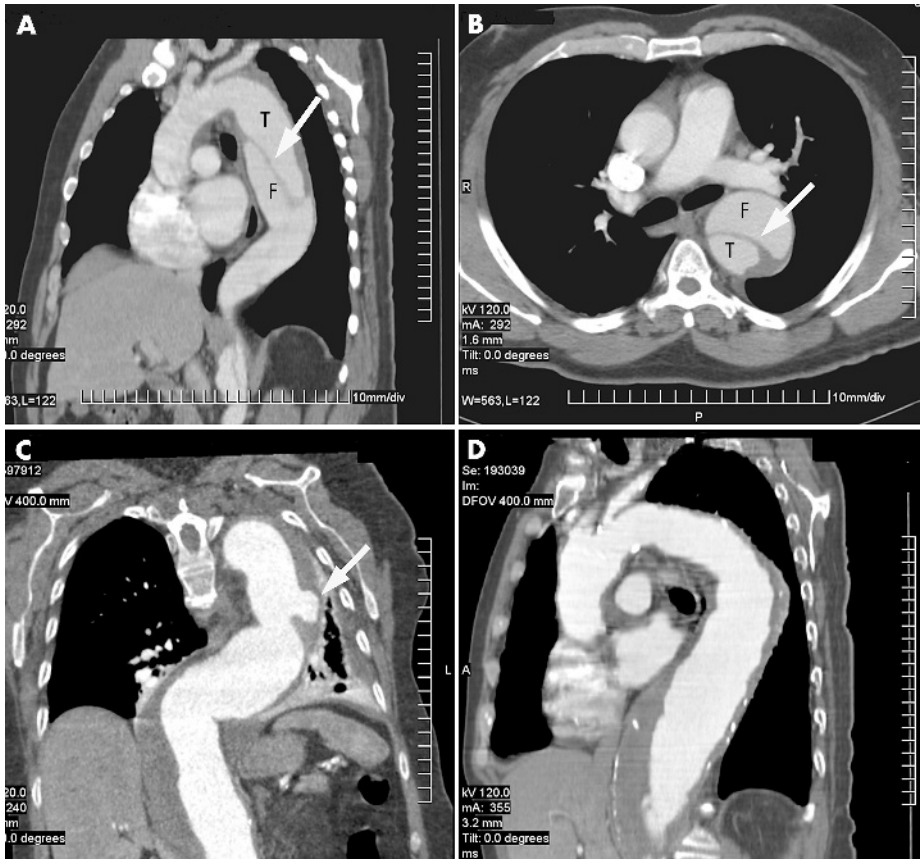

Figure 2 Examples of thoracic aortic pathology on CT. (A) Parasagittal image showing type B aortic dissection flap (arrow). T, true lumen, F, false lumen, LSA, left subclavian artery. (B) Axial image showing the same dissection as (A) in the descending aorta. (C) Parasagittal image of PAU in the descending aorta (arrow). (D) Parasagittal image of a descending thoracic aneurysm. Maximal diameter about 8 cm.

AAS (64%), particularly so when pathology is confined to the ascending aorta (47%).[14]

Once AAS is suspected on the basis of clinical presentation, particularly once myocardial infarction has been excluded, contrast enhanced computed tomography (CT) is considered the diagnostic test of choice. It is widely available, permits assessment of other thoracic structures, and provides 3D reconstructed images that are essential in planning surgery or interventional procedures.[11] Its sensitivity for dissection and IMH approaches 100%.[15]

Other imaging modalities include magnetic resonance imaging (MRI) and transoesophageal echocardiography. MRI, although providing greater anatomical detail than CT, is limited by expense, availability, and the need for MR compatible monitoring equipment.[4 16] Echocardiography, although an effective portable diagnostic tool,[1] does not visualise the aortic arch or abdominal aorta well or permit 3D reconstructions. In reality all modalities have their proponents and their advantages and limitations have been reviewed in detail.[7]

TREATMENT OF ACUTE AORTIC SYNDROME

Figure 3 provides a potential algorithm for the diagnosis and treatment of AAS. Once identified initial management entails pain relief and aggressive maintenance of systolic blood pressure between 100 mm Hg and 120 mm Hg to avoid further extension or rupture. This is most commonly achieved using β blockers.[4 16] The early advice of and transfer to a regional vascular surgical unit is essential. Facilities here would include an interventional suite with theatre specifications and a suitably broad range of stent grafts in stock. A collaborative approach between interventional radiologists, vascular surgeons, and intensivists is required. Further management is dictated by the site of the lesion, whether the patient remains symptomatic (for example, with persistent aortic pain or end organ ischaemia) and if there is evidence of progression on serial imaging.

Treatment of type A (ascending aortic) pathology

Dissection, IMH, or PAU located in the ascending aorta (type A disease) is a strong indicator of disease progression. Acute type A dissection has a mortality of 1%–2% per hour during the first 24–48 hours of presentation. It can rapidly extend towards the heart and cause death by tamponade or progress more distally to occlude arch vessels. Type A IMH, PAU, and thoracic aneurysm are likewise at increased risk of complications. Medical management alone is ineffective. Endovascular treatment although reported in highly selected patients for the primary treatment of type A dissection[17 18] is often not possible because of the anatomical restrictions of securing a stent graft within the ascending aorta. Therefore, early surgical intervention is advocated for type A pathology[3 5 6 8 10 19 20] and this has been shown to improve prognosis.[21–25]

Treatment of type B (descending aortic) pathology

A conservative medical approach to type B aortic disease is considered acceptable when the lesion is stable. Specifically the patient should be asymptomatic, complications such as end organ ischaemia should be absent, and the lesion should be limited in size with no progression on serial imaging.[8] This has long been the therapeutic strategy for aortic dissection and more recently has been applied to IMH and PAU.[26 27] The challenge however arises in identifying those lesions that are truly stable and how to manage lesions that are unstable.

Despite the perceived benign nature of type B lesions there is a significant risk of rupture. The rupture rate for type B dissection is 4%–8%[8 28] and for IMH and PAU it is as high as 35%–50%.[5 29] A number of prognostic indicators have been

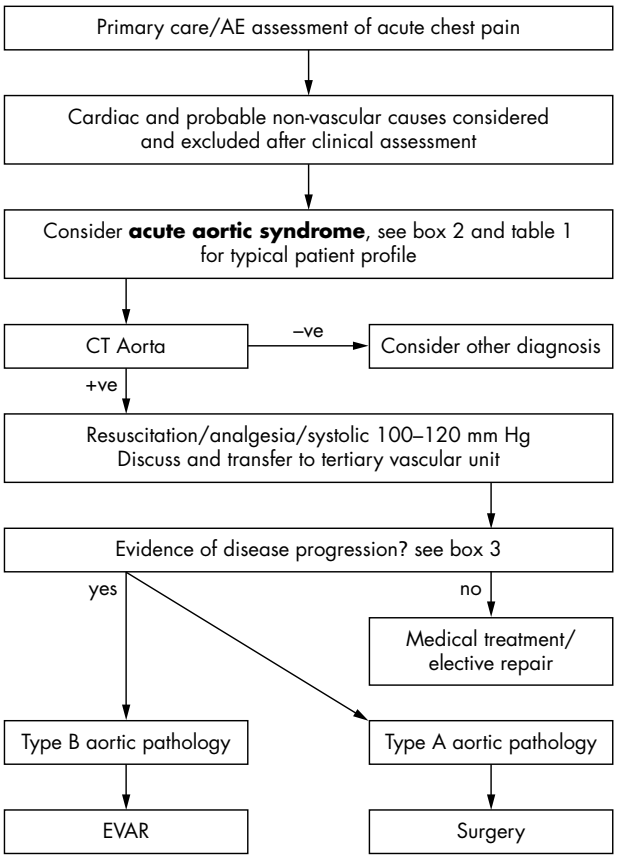

Figure 3 A diagnostic and treatment pathway for acute aortic syndrome presenting as acute chest pain.

identified and these are listed in box 3. These broadly include persistent clinical symptoms and an increase in the size or depth of a lesion. A further important situation in which progression is recognised is when IMH has formed secondary to a PAU.[20] The presence of ulcer-like projections in the area of an IMH is much more likely in the descending aorta and therefore identifies a high risk group of patients with type B disease.[30] Even with close monitoring of these poor prognostic indicators a significant proportion will go on to rupture unpredictably, suggesting the natural history of these lesions is complex. In the presence of any of these factors, clinical or radiological, urgent intervention is required to avoid rupture. The traditional approach to this has been open surgery.[31]

Open surgery for acute type B aortic lesions

There are a number of factors that make open surgery unsatisfactory for the treatment of type B pathology. Many patients at greatest risk of rupture are paradoxically poor surgical candidates being typically elderly with complex comorbidity (see table 1). Open surgery for symptomatic thoracic aortic disease therefore carries high mortality (33%–64%),[6 32 33] which increases when there is associated renal or mesenteric ischaemia (50%–87%).[5]

Surgery is also complicated by serious morbidity.[33] These include cardiac (10%) and respiratory complications (28%),[34] renal failure (16%–17%),[34 35] and paraplegia (7%–27%). Paraplegia is a particularly devastating complication and occurs because of interruption of the intercostal blood supply to the spinal cord by surgical graft material.[34–37] Therefore although surgical treatment has been used in cases that are unsuitable for conservative management, it has failed to improve prognosis and is in fact no better than medical therapy.[3 22 38]

Box 3 Indicators of aortic disease progression suggesting a need for early intervention[6 8 20 29]

- Persisting acute aortic pain despite medical management
- Increasing aortic wall thickness or diameter
- PAU greater than 20 mm in diameter or 10 mm in depth
- Increased volume or extent of haematoma
- Bulging haematoma
- Extra-adventitial blood
- Increasing pleural effusion
- IMH associated with PAU

Endovascular aortic repair (EVAR)

The first endovascular (thoracic) aortic repairs (EVAR) were reported in 1994.[39] These use stent grafts, which because of their size, are normally introduced through a femoral cut down either within a sterile angiographic suite or operating theatre. A stent graft, as the name implies, consists of two main components. A self expanding metallic stent or frame secures the device in the vessel and provides a seal, which excludes periprosthetic blood flow—that is, blood flow between the outside of the stent graft and diseased vessel wall (endoleak). The stent also acts as a support for the graft material that is analogous to the surgical graft used in open repair and is made of materials such as dacron and polytetrafluoroethylene (PTFE).[3] Stent grafts therefore exclude the diseased segment of aorta. An example of such a repair is shown in figure 4.

There are a number of potential benefits in the use of endovascular techniques to treat aortic pathology. These are summarised in box 4 and detailed below.

Benefits of EVAR in elderly patients

EVAR is a minimally invasive technique. This permits the procedure to be undertaken under a local or regional anaesthesia in surgically unfit patients.[22 39 41 42] An added benefit of this is that patients may be monitored during the procedure for distal neurological function.[32] Both repair time (in one study taking only 1.6 hours compared with eight hours for surgery[22]) and blood loss are also reduced; important factors in unfit and haemodynamically unstable patients.[43 44] EVAR also avoids the need for thoracotomy, single lung ventilation, full heparinisation, bypass, and cross clamping.[42 45] These are all important factors accounting for morbidity and mortality related to surgery.[22 46] Patients therefore recover more rapidly after the procedure and studies have shown impressive ITU and hospital stays of 1.7–2.4 and three to eight days respectively.[9 47 48] Significant financial savings could therefore also result.[22]

Lesion specific benefits of EVAR

Within the context of dissection the presence of a stent graft has a number of beneficial effects. Sealing the proximal entry tear to an aortic dissection decompresses the false lumen.[3 16] This results in consolidation or thrombosis of the false lumen, which is a good prognostic indicator.[17] Eventually the false lumen decreases in size because of retraction of thrombus.[49–52] Reforming the collapsed true lumen also decreases the risk of aneurysm formation[4 22] and has been found in 79% of patients on follow up.[17] A further benefit of stenting the dissection is revascularisation of occluded aortic branches. This reverses end organ ischaemia and has been reported to occur in 76% of patients.[17]

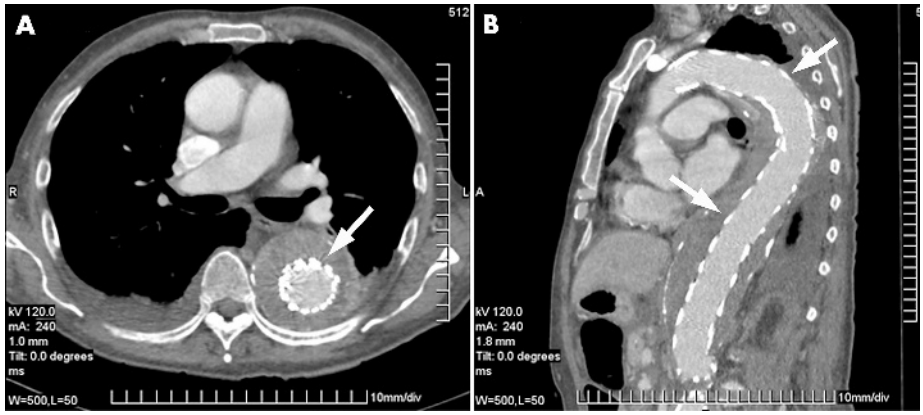

Figure 4 (A) Axial and (B) parasagittal CT images of an endovascular stent graft repair (arrows) of the descending thoracic aneurysm shown in figure 2D.

There is now also a growing interest in treating IMH and PAU using these endovascular techniques as their focal nature makes them ideal lesions for stent graft repair. As discussed they most commonly occur in the descending thoracic aorta (90% of PAU and 71% of IMH[8]) and have a particularly aggressive natural history with a much higher risk of rupture compared with dissection.[8 12] Patients are also typically older than those with aortic dissection and they are more likely to have concomitant atherosclerotic disease. In these lesions stent graft repair reduces wall stress and therefore prevents progression of the diseased aortic segment to aneurysm formation or rupture.

The evidence for endovascular aortic repair in AAS

There is a growing body of evidence to support the use of stent grafts in AAS. A number of case reports and studies involving small series of patients presenting with AAS show implantation of the stent graft with a success rate between 96% and 100%.[17 48 53–59]

Mortality rates are also impressive. In a number of small studies, hospital and 30 day mortality rates have been reported between 0% and 16.7% for acute thoracic aortic pathology, including type B rupture and anuerysmal disease.[9 22 41 42 45]

Collectively these studies report almost no serious morbidity including paraplegia.

A larger study of 120 patients with type B dissection, four with IMH and 15 with PAU supports these findings. It reported 98% successful implantation in the dissection group with 1.7% mortality and 100% success in the later two groups with no mortality at one year and no neurological complications.[3] In addition a literature review of 641 patients including both elective and acute EVAR of thoracic aortic dissections and aneurysms found an overall mortality of 6.2% with a 2.4% mortality rate for that particular centre.[60]

There is further encouraging evidence for the use of EVAR in treating patients with PAU and IMH. A study involving 14 patients with type B pathology including PAU (all but one patient with contraindications to surgery) achieved successful stent graft placement in all.[55] Mid-term follow up of 26 elderly patients, over half of whom were unfit for surgery, estimated 85% one year and 70% five year survival after endovascular repair of type B PAU.[61] The successful treatment of acute aortic rupture caused by PAU has also been described with successful follow up at one year.[54] Similarly a small study of type B IMH presenting as AAS has shown the successful emergency placement of stent grafts with symptom free follow up and no endoleak at 18 months.[62]

Further studies have attempted to compare more directly EVAR with surgery. Twelve patients with sub-acute type B aortic dissection when compared against 12 matched controls undergoing surgery, found no morbidity or mortality in the endovascular group. This was in contrast with 33% mortality and 42% serious morbidity after surgery, both statistically significant.[22] A further study of 54 patients with thoracic aneurysms, type B dissections, and traumatic ruptures managed endovasculary (26 patients) and surgically (28 patients) found the immediate outcome to be better in the stent graft group. These patients had lower rates of mortality

Box 4 The potential benefits of EVAR in the treatment of type B aortic pathology

Procedural benefits

- Minimal access procedure
- Procedure undertaken in surgically unfit patients
- Short procedure time
- Minimal blood loss
- Shorter recovery time
- Financial savings

Technical and clinical benefits

- Obliteration of false lumen in aortic dissection
- Occlusion of diseased segment of aorta
- Reversal of end organ ischaemia
- Reduced morbidity and mortality compared with surgery

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(3.8% *v* 17.8%), paraplegia (0% *v* 3.6%), renal failure (3.8% *v* 14.3%), and ventilation requirements (7.7% *v* 28.6%).[18] A literature review combining a number of studies confirms these lower rates of mortality and serious morbidity for EVAR when compared with emergency surgery for type B dissection.[3]

Current limitations of endovascular aortic repair

Despite these impressive results limitations to the use of EVAR are also recognised.

In particular it requires favourable anatomy.[40] There must be a comparatively normal section of aorta at the distal and proximal limits of the diseased segment, usually more than 15–20 mm in length.[63] This allows the stent graft to be fixed in position and provides a reliable seal.[16] Disease affecting the great vessels, excessive thrombus, calcification, and vessel tortuosity may also limit its use. In practice however, even using the strictest criteria a large proportion of patients are still suitable, including those in whom a general anaesthetic for surgery would be contraindicated.[43]

Endovascular repair may also result in arterial damage at the site of access because of a combination of a large delivery system and local atheroma.[32] Bowel infarction and distal embolisation are more serious complications and although their incidence is difficult to assess from the literature, it is unlikely to be greater than surgical repair. Clearly EVAR also requires the support of a dedicated unit.

A recognised late complication of abdominal aortic stent grafts is structural failure. It is unclear from the available studies whether thoracic EVAR will be similarly affected.[33] Potential stent graft failures include fracture, migration, erosion, infection, and endoleakage.[40] Endoleak has been reported with an incidence as high as 11%.[45] It can occur because of stent graft migration and resulting reperfusion of the aneurysm sac can cause rupture after aneurysm repair.[64] Endoleaks may require additional stent grafts to be inserted. Despite this EVAR still represents a risk reduction for rupture of 97%[65] and the impact of new generation stent grafts is yet to be determined.

CONCLUSION

The evidence for EVAR in patients presenting with AAS is encouraging. However, it should also be interpreted with some caution. Most studies are limited to case reports and small single centre series.[40] These provide only short to mid-term follow up in contrast with well established surgical series.[45] Several studies have therefore called for the support of long term follow up data.[3] Despite these concerns current evidence may in fact underestimate the true worth of EVAR. Most studies involve patients who are poor surgical candidates. However, as with surgery, good surgical candidates have significantly better outcomes with stent grafting than those with pre-procedural morbidity.[66]

There are a number of reasons for the currently limited evidence base. At present, within single centres the number of patients undergoing treatment is comparatively small. Data could be collected over a longer period to produce a more substantial longitudinal series; however, this is less meaningful with rapid changes to technique and technology. Prospective randomised trials are also lacking from the literature. Although these would be ideal in evaluating the role of stent grafts in AAS[3] they could now also prove unethical in view of poor surgical outcomes and the current favourable evidence towards stent grafting.[32] An alternative approach is currently underway with submission of data to the National Thoracic Stent Graft Registry.[33] This register is now under the auspices of the National Institute for Health and Clinical Excellence (NICE).

Although a lack of long term follow up does raise questions over the durability of stent grafts, this may have less significance in a large proportion of patients who are elderly with complex comorbidity.[42] In addition there is every reason to be optimistic regarding the efficacy of EVAR. Technology is constantly improving with more flexible stent grafts, smaller delivery systems, and better deployment mechanisms being developed.[32] Workers are also becoming more experienced in techniques and patient selection. This has in fact led to wider clinical applications. EVAR has been used as a holding procedure before definitive surgical treatment in cases of end organ ischaemia[16] and complex polytrauma.[47] In addition endovascular revascularisation of viscera and limbs is particularly beneficial for patients with acute type A pathology with surgical repair being undertaken once the ischaemic injury has resolved. This is related to a better outcome as post-procedural mortality seems to correlate with the degree and duration of ischaemia before intervention.[5]

As a result of its current success EVAR also faces a number of challenges. There is currently a shortage of skilled interventionalists within the United Kingdom and an increasing demand on imaging services is also probable. This not only applies to requests for diagnostic imaging but also to potentially lifelong periodic surveillance scans to assess for stent graft failure.[39] Finally, there are cost implications to maintaining a suitably broad stock of stent grafts and this can lead to delays in treatment.[67]

In conclusion, AAS merits greater awareness. This will encourage the development of effective and efficient diagnostic and treatment pathways for a number of related aortic conditions. EVAR is now a viable alternative to surgical repair in high risk patients with type B acute aortic syndrome pathology. Short and mid-term evidence has shown significantly lower rates of mortality and morbidity and this is reflected in recently published NICE guidance, which recommends EVAR for thoracic aortic aneurysm and dissections as a ''suitable alternative to surgery in appropriately selected patients''.[68] Long term results are now awaited to assess its true efficacy.

QUESTIONS (TRUE/FALSE); ANSWERS AT THE END OF THE REFERENCES

1. Patients presenting with acute aortic syndrome are typically young trauma patients.
2. The chest radiograph is a useful screening tool for acute thoracic aortic pathology in the emergency department.
3. Endovascular aortic repair (EVAR) can be used to treat symptomatic Type B thoracic aortic pathology.
4. Paraplegia is a potential risk of surgical or endovascular thoracic aortic repair.
5. Endoleak complication occurs because of perforation of the endovascular stent graft.

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ANSWERS

False. Patients are typically elderly with complex co-morbidity making them poor surgical candidates. 2. False. This has a poor sensitivity. CT is currently the imaging modality of choice in most departments. 3. True. Type A (ascending thoracic aortic) disease usually requires surgical intervention and medical intervention is reserved for non-symptomatic and stable type B disease. 4. True. Paraplegia occurs because of occlusion of intercostals vessels to the spinal cord but evidence suggests it is much less common with endovascular repair. 5. False. Endoleak is attributable to an inadequate seal between stent graft and vessel wall resulting in flow of blood between the two. It is associated with an increased risk of aortic rupture.

WEB TRAWL

This month's web trawl examines three very different web sites that give information on Alzheimer's disease and other forms of dementia.

http://www.dsdcengland.org.uk This is a communal web site for England's eight Dementia Service Development Centres (DSDCs), which provide services and information about dementia for a defined geographical area. From the home page, the user may access pages for each of the eight centres (each page contains a statement pertaining to the work of the centre, along with contact details). Links are also provided to a series of pages giving details of research projects undertaken by the various DSDCs, seminars, and other similar events. It does appear some time however since these pages were updated, and some of the information, particularly with regard to the seminars is out of date. Although the web site covers the eight English DSDCs, there are also links provided to the equivalent web sites in Scotland, Wales, and the Republic of Ireland. Overall, the information provide on this web site is very specialised and would be largely of interest only to those who are providing services for the care of patients with dementia.

http://www.dementia.stir.ac.uk This is the web site of Scotland's Dementia Services Development Centre. The centre provides information on dementia, as well as developing and disseminating research into the condition. From the home page, the user may navigate to a series of pages detailing education, training, and other services the centre provides, along with lists of publication available for purchase. The web site is aimed largely at those working with, and caring for, patients with dementia and would be of value not only to medical and nursing staff, but also social workers and staff of day centres and care homes. The web site does not contain a great deal of information that would be directly relevant to patients and their families, who would be better directed to the web site of the Alzheimer's Society.

http://www.alzheimers.org.uk This is the web site of the Alzheimer's Society; the UK based charity for patients with Alzheimer's disease, their family, and carers. The home page is clearly laid out and contains numerous (more than 50) links to pages covering subjects ranging from basic facts about the disease and its diagnosis to advice for carers and suggestions for fundraising. A ''news'' section, also accessed from the home page, is regularly updated, with the most recent item having been posted one day before the writing of this review. Much of the information is aimed at patients and carers, but health professionals are catered for also. Details are provided of a variety of training courses, along with information about drugs that are now used in the treatment of Alzheimer's disease, the latter being available for download in pdf format. Families, carers, and patients themselves often have many questions about this distressing condition. This web site contains a wealth of material, and is likely to provide much of the information they may be searching for. This site, therefore, is well worth recommending to patients and their families.

Robyn Webber
Web Editor